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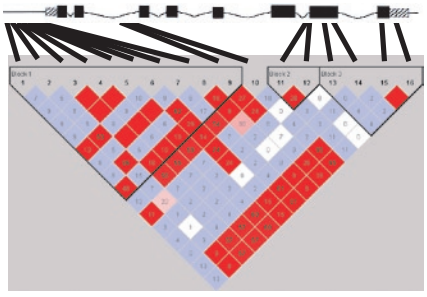
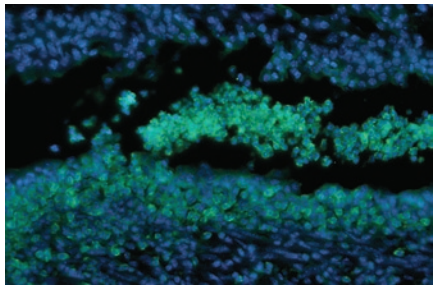
Chromogranin A polymorphisms in hypertension

Chromogranin A (CHGA) is a protein stored in the dense core vesicles of chromaffin cells, which contain catecholamines. The protein is cleaved to produce a number of peptides, several of which are biologically active, including vasodilators. One of its peptide products is catestatin, a peptide that inhibits release of catecholamines. As they report in this issue, Chen *et al.* studied common genetic variants in the promoter of chromogranin A in a large population. They found that one haplotype (haplotype B) blunted blood pressure response to cold stress. Using cells transfected with a reporter attached to the chromogranin haplotypes, they found that haplotype B decreased expression of the reporter as compared with the other major haplotype, haplotype A. Given that some peptides released from chromogranin processing possibly inhibit catecholamine release, these results suggest that common genetic variants in the CHGA promoter



may regulate heritable changes in blood pressure. **See page 115.**

CXCR2 and urinary tract infections



Infection with uropathogenic *Escherichia coli* results in migration of neutrophils into the kidney interstitium and rapid clearance of the infection. Previous studies have shown that mice lacking the chemokine receptor CXCR2 were unable to clear experimental pyelonephritis. However, because this receptor is expressed both on neutrophils and on the surface of renal epithelial cells, the question arises of which of these cells controlled the clearance of bacteria. Svensson *et al.* present new studies that distinguish between the contribution of neutrophils and that of epithelial cells to this phenomenon. Mice lacking CXCR2 in the epithelial cells but reconstituted with bone marrow from wild-type mice readily cleared the induced infections, indicating that CXCR2 on neutrophil membranes was critical in this response. Mice that had CXCR2 only in their epithelial cells had a transient chemokine response, while mice lacking the receptor in their neutrophils were unable to recruit neutrophils and thus could not clear the bacteria. These studies provide compelling evidence for the role of the CXCR2 receptor in attracting neutrophils to the kidney and in clearance of bacteria. **See page 81.**

Acute renal failure in CKD

Acute renal failure acquired during a hospital stay is common, but does preadmission chronic kidney disease (CKD) increase its risk or severity? Although this might seem an obvious question, little systematic research on the subject has been done. Hsu *et al.* conducted a detailed analysis of patients enrolled in Kaiser Permanente hospitals of northern California. They compared 1,746 patients who required dialysis for acute renal failure acquired during their hospital stay with the more than 600,000 who did not require dialysis. All patients had preadmission measurements of plasma creatinine levels, which were used to estimate their glomerular filtration rates. The authors found that patients who had stages 3 to 5 CKD had a much higher risk of acquiring acute renal failure following hospital admission. Other independent risk factors for acute renal failure were preadmission diabetes, hypertension, and proteinuria. **See page 101.**